Foreign Animal Disease Report

United States Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services

Emergency Programs

Number 15-3 Fall 1987

In This Issue

Current Events

Emergency Field Operations

On April 7, 1987, a Colorado veterinarian collected suspicious larvae from a neck wound on a dog. The owner and the dog had recently returned from a trip to Venezuela. They reentered the United States through Miami International Airport, flew to Albuquerque, New Mexico, where they spent the night, and returned to their home in Pagosa Springs, Colorado. Preserved larvae were sent to the National Veterinary Services Laboratories (NVSL) in Ames, Iowa, where on April 22, 1987, they were identified as mature screwworms (Cochliomyia hominivorax). The area in Colorado where the dog was located is considered too cold for the flies to survive; however, they could have survived in the Albuquerque and Miami areas. Task forces were immediately established in the States of New Mexico and Florida. Sterile fly releases from the ground and from airplanes were begun and continued twice weekly for 6 weeks, along with active surveillance in a 50-mile radius of sites where the dog had stayed. Surveillance continues in both areas. No screwworms have been found in the surveillance areas or elsewhere in this country. (See screwworm review, 11-2:7-11.)

On June 19, 1987, vesicular stomatitis was diagnosed by virus isolation from three feral swine on Ossabaw Island off the coast of Georgia. (See 11-4:1-2.) Studies by NVSL revealed the virus to be New Jersey type. Nucleic acid fingerprinting confirmed that this virus was the same strain found on the island in 1983. The State of Georgia issued a quarantine which was in effect for 30 days after the last clinical case was detected.
(Dr. A. G. Grow, 303 436-8066)

READI System Update

The Recorded Emergency Animal Disease Information (READI) system is now on PRIME hardware. (See 14-4:3.) READI computer software has been greatly enhanced to be more user friendly and supportive at every operational level. Training at Veterinary Services (VS) regional offices has been completed. All area offices will be on-line by the end of September.

VS field investigation forms 12-27, 12-27A, and 12-27B have been modified and improved to work with the new READI System. Epidemiological information not previously available can be entered into the system and retrieved in a variety of different formats, as needed. The new forms were distributed to the field and used in a simulated national animal disease emergency during August. (Dr. E. I. Pilchard, 301 436-5959 and Dr. A. G. Grow, 301 436-8066)

World Animal Disease Update

Chile continues to report outbreaks of foot-and-mouth disease (FMD) type O_1. The first outbreak since May 1984 was in February 1987. By July 10, 1987, 120 separate outbreaks were recognized, and an estimated total of 29,000 animals were destroyed. Control measures adopted by Chilean authorities included epidemiological investigations, quarantine, destruction of animals, and disinfection of affected premises. The Chilean Government authorized indemnity funds of 800 million pesos ($3.7 million) to assist affected cattlemen.

Rio de La Plata Basin countries (Argentina, Uruguay, and Rio Grande do Sul, Brazil) report that a mutant of FMD virus type A is causing a wave of outbreaks of FMD. The new mutant has been classified as A_{81} and is now being added to the tetravalent vaccine containing types A_{79}, O_1, $C_{3(84)}$, and $C_{3(85)}$. The Panaftosa office of the Pan American Health Organization, Rio de Janeiro, Brazil, is coordinating efforts to increase quarantine areas, halt cattle movement, disinfect affected areas, and ring vaccinate. Prior to April 1987, subtype C_3 was the main FMD virus affecting cattle in Argentina.

During May 1987, Colombia reported an outbreak of type A FMD in Area III of the FMD control zone. (See 11:1 for description of Area III.) The rest of Colombia, outside of the FMD control area, reported 107 outbreaks of FMD types A and O from January through May 1987.

In Europe, Italy continues to report outbreaks of FMD type A_5 virus. By the end of March, 139 outbreaks had been reported during 1987.

In the Near East, Kuwait reported one outbreak of FMD type O. Also, after having had no outbreaks of FMD since 1981, Israel reported an outbreak of type O virus in two neighboring flocks of sheep.

In Southeast Africa, Zimbabwe and Tanzania reported outbreaks of FMD types SAT_2 and SAT_1, respectively.

The world reference laboratory for FMD at Pirbright, England, confirmed FMD virus types A and O from Saudi Arabia; type O from Bahrain, Hong Kong, and Syria; type A from Cameroon and Thailand; and types A and Asia I from Bangladesh.

Belgium was declared free of African swine fever (ASF) and FMD by the Office International des Epizooties on March 19, 1987. However, Belgium reported 23 outbreaks of hog cholera (HC) through March 1987. There were 7 outbreaks of HC in Italy, 4 in

Yugoslavia, 13 in the Federal Republic of Germany, and 1 in the Netherlands. Hog cholera was previously reported in the Netherlands in March 1986.
The Netherlands reported their first case of **contagious equine metritis** (CEM) on July 22, 1987.

Portugal reported 50 outbreaks of **contagious bovine pleuropneumonia** (CBP) in January and February. Kuwait reported 59 outbreaks during the same period. In Africa, Angola, Mali, Namibia, and Benin also reported CBP.

Ireland confirmed **avian influenza** (AI) on a turkey farm on February 11, 1987. A quarantine was immediately placed on farms within a 5-mile radius, and birds on the infected farm were slaughtered. All poultry flocks in the infected area were serologically tested and found to be free of AI antibodies. The origin of the outbreak is unknown.

Fiji has been added to the list of countries declared free of **viscerotropic velogenic Newcastle disease, hog cholera,** and **swine vesicular disease.** This action relieves restrictions on the importation from Fiji into the United States of swine, pork, pork products, carcasses and parts, and products of poultry and birds. (Dr. Percy W. Hawkes, 301 436-8285)

245

Laboratory Diagnosis of Hog Cholera //

Although the United States is free of HC, the disease remains endemic in the swine population of much of the rest of the world. In the Western Hemisphere, only Canada and the United States are free of HC. The risk of introduction is, therefore, very great and justifies the surveillance system that is currently in use. Greatest risk is incurred by the illegal introduction of infected swine into Texas from Mexico. Despite the manufacture and sale of many types of vaccines, HC is a major health problem for Mexican swine producers. Clinical cases frequently occur in the Mexican States bordering the United States. The other major risk for the introduction of HC is the feeding of commercial garbage to swine, a practice most prevalent along the eastern seaboard of our country. Feeding uncooked table scraps of pork or illegally imported fresh or poorly cured pork products to garbage-fed swine is considered the second most likely means of introduction of HC.

Accordingly, the U.S. Department of Agriculture's HC surveillance program is focused on Texas and the major garbage-feeding States of New Jersey, Massachusetts, Rhode Island, and New Hampshire.

Sera from slaughter swine raised along the Rio Grande River and around the major cities of Philadelphia, New York, and Boston are randomly sampled at the rate of 500 to 700 sera per month, or 6,000 to 10,000 sera per year, and are submitted to the National Veterinary Services Laboratories (NVSL) for HC serological testing. In addition, tissue and serum specimens are submitted to NVSL by Foreign Animal Disease Diagnosticians from field investigations of suspected swine diseases.

For HC serology, a fluorescent antibody neutralization test is performed by screening all survey sera at the 1:16 dilution against strain A HC virus. Swine positive by that assay usually

have bovine virus diarrhea (BVD) antibodies that cross-react with HC virus. To determine whether the screen test reaction is specific, positive sera are serially diluted and tested against both viruses. Most commonly, swine previously exposed to BVD virus will have BVD titers that are 4- to 16-fold greater than the HC titers, i.e., 1:256 BVD and 1:16 titer against HC. Neutralizing titers that are equivalent for both viruses or higher for HC are reported by telephone to the National Field Operations Staff and usually result in an investigation of the owner's premises.

HC viral antigen can be detected in tonsil biopsies. Specimens of tonsil, spleen, and lymph node obtained at necropsy are tested using the fluorescent antibody tissue section test (FATST). Results from the FATST can be available in as little as 3 hours. However, a FATST positive test result from a suspected HC case must be finally confirmed by the isolation of HC virus. Virus isolation requires from 24 to 72 hours after tissues are received at the laboratory. If the virus isolation assays are negative, specimens from additional swine may be needed to obtain a definitive diagnosis.

Newer diagnostic assays are on the horizon for the laboratory diagnosis of HC. Monoclonal antibodies produced against HC and BVD viruses hold great promise for the development of reagents that will identify HC virus antigens in tissues as a definitive one-step process, and will provide for direct differentiation of specific HC antibodies from BVD antibodies. The NVSL is beginning to evaluate these new tools now. (Dr. G. A. Erickson, 515 239-8551)

Focus on... Duck Plague

Duck plague (DP, duck virus enteritis, eendenpest, peste du canard, or entenpest) is a contagious disease of Anseriformes caused by a herpes virus.

The disease was first recognized in the Netherlands and was reported by Baudet in 1923. The threat of DP to commercial duck raising in North America was realized by 1967 when the disease struck duck farms at the east end of Long Island, New York.

Laboratory confirmation of DP at the U.S. Department of Agriculture's Plum Island Animal Disease Center (PIADC) in 1967 was followed by an in-depth study of the disease. The disease was then renamed "duck virus enteritis" to avoid confusion with fowl plague (avian influenza) and reduce the likelihood that such confusion could adversely affect commercial sales of Long Island ducks.

Viral Properties

The virus of DPV is a herpesvirus: Duck herpesvirus 1 (Anatid herpesvirus). Dutch workers attenuated the virus by serial passage in embryonated chicken eggs. The attenuated virus retained its antigenicity and immunogenicity but was not pathogenic to ducks.

In electron micrographs of infected cell cultures, DP nuclear particles were approximately 91 nm in diameter, with core structures approximately 32 nm in diameter. Larger, cytoplasmic particles were approximately 191 nm in diameter, with densely stained cores of 75 nm. Duck plague virus (DPV) is non-hemagglutinating and non-hemadsorbing. The virus causes Cowdry type A intranuclear inclusion bodies in infected chicken and duck embryo cell cultures. The plaque forming ability of the virus in cell culture has been demonstrated.

Field strains of DPV are of identical immunological type. Isolates from ducks originating in different geographical locations in the United States and the Netherlands demonstrated an identical serological and immunogenic relationship.

Duck plague virulent and attenuated viruses are sensitive to ether and chloroform. Their infectivity is destroyed after 2 hours at $50^{\circ}C$, or after 10 minutes at 60°. At $22^{\circ}C$, infectivity declines slowly, and the virus is not infective after 30 days. Also, DPV strains are inactivated immediately at pH 11 and at pH 3 or lower. Duck plague chicken embryo adapted virus is inactivated after 15 seconds by a 1:256 dilution of One Stroke Environ disinfectant at $37^{\circ}C$. (Vestal Laboratory, Div. of Chemed. Corp., St. Louis, Missouri 63110, U.S.A.*)

Differences in virulence between DPV isolates have been reported. These differences can be eliminated by 1 to 3 passages in embryonated duck eggs or 1- to 3-day-old ducklings.

Virus Survival

Virulent DPV persisted in the laboratory in filtered and unfiltered lake water for at least 2 months. Although it is

*Disclaimer: Mention of a trade name is for information only and does not constitute endorsement of the product by the U.S. Department of Agriculture or recommendation over other products which may be similarly effective.

sometimes assumed that virulent DPV in feces and other bodily discharges is diluted to harmless levels when deposited in water, infectivity may persist in water at low temperatures.

Vaccine

Passage of DPV in chicken embryo and chicken fibroblast cell cultures attenuated the virus. Twenty serial passages of DPV in chicken embryos, conducted by the Dutch scientists, resulted in a strain which was lethal to chicken embryos but not to ducks. At PIADC, the viral strain from the Netherlands was propagated in cell culture, plaque cloned three times, and propagated in specific pathogen-free 8-day-old chicken embryos. After determination of freedom from contaminants and the establishment of the immunogenic dose, seed virus in the form of chorio-allantoic fluid was released from PIADC. This attenuated virus is presently used in the United States for vaccine purposes.

In addition, the virulent DPV isolated from the ducks during the previously mentioned outbreak on Long Island was successfully adapted to cell culture. The resultant attenuated virus protected ducks against DPV infection.

Results of limited vaccination trials with inactivated preparations were inconclusive. Protection of ducks as early as first and fourth day after vaccination was reported to be attributed to an interference phenomenon. There was no correlation between the humoral antibody produced by the inactivated vaccine and protection.

Geographical Distribution

Epizootics of DP have been reported from the Netherlands, Belgium, Canada, China, England, France, India, and Thailand. DP appeared on Long Island, New York, in January 1967. Subsequent outbreaks of the disease were limited to domestic duck flocks in New York, Pennsylvania, and Maryland. It was then recognized in California in 1972. A major DP epizootic among free-flying wild waterfowl occurred in January 1973 at Lake Andes, South Dakota. An estimated 42 percent of 100,000 mallards and 3 percent of 9,000 Canadian geese died after entering the area. Small numbers of black ducks, pintail-mallard hybrids, American widgeons, wood ducks, common goldeneyes, redheads, and common mergansers also died. DP has appeared occasionally since 1973 in the Eastern, Midwestern, and Western States.

Viral Hosts

The family Anatidae (ducks, geese, and swans) of the order Anseriformes are susceptible to natural infection by DPV. The number of water fowl species incriminated in DP epizootics is extensive. Mallard ducks have been reported as reservoirs of infection.

The mature chicken is resistant to duck plague. However, chickens inoculated with DPV intracerebrally, between the ages of 1 day and 2 weeks, are susceptible. Adult pigeons, rabbits, guinea pigs, and mice are not susceptible to experimental infection. Duck plague has not been reported in humans.

Transmission

Duck plague is disseminated by direct and indirect means. DPV

can be transmitted by oral, nasal, and cloacal routes. The primary mode of transmission is exposure of susceptible waterfowl to a DPV-contaminated environment. Contaminated water plays a principal role in transmission and spread of DPV when ducks are raised in an aquatic environment. The infection can be maintained by infected birds and contaminated fomites in the absence of virally contaminated water. Introduction of contaminated feed and of apparently normal flying waterfowl are responsible for disease dissemination. The infection has been established indirectly by contaminated feed sacks, trucks, and marketing crates.

Reported serological results do not suggest that duck plague is an important disease in wild ducks and geese. However, the greatest reported incidence of duck plague in wild Anseriformes occurred during their normal migratory periods, suggesting that susceptible birds were moving into a DPV-contaminated environment.

On Long Island, domesticated ducklings are progressively moved as they grow and are housed in premises previously occupied by older birds. This practice has caused an increased incidence of DP among market ducklings.

There is experimental evidence that DPV can be transmitted by waterfowl in which the infection is dormant. The virus has been isolated from cloacal swab specimens from normal-appearing waterfowl 17 and 45 days after recovery from an experimental infection. The waterfowl had high levels of circulating virus neutralization antibodies. It appears possible that similar inapparent infections occur in free flying waterfowl. Such waterfowl could transmit the virus by contaminating surface waters used by domestic ducks.

Suprainfection and mortality of DPV carriers was recently reported. The immune mechanisms involved in DPV suprainfection are unclear. It is of epizoolotical value to be aware that birds surviving an initial DPV infection are not necessarily protected from death due to a second DPV infection.

Pathogenesis

The incubation period of DP ranges from 5 to 12 days followed by a course of 4 to 10 days. The incubation period in infection by contact is longer than that following parenteral infection.

Ducks of all ages are susceptible to DPV. Infected waterfowl become listless, sit most of the time with drooping wings, and crawl in order to move on land. They are reluctant to swim. The feathers are ruffled. Lacrimation is excessive, forming a wet ring of feathers around the eyes. The conjunctivae become congested, reddened, and edematous. As the disease progresses the eyelids stick together. Nasal discharges increase, vocal sounds become hoarse, and respiration is labored. There is watery diarrhea, with soiled feathers around the cloaca, and congestion of the cloacal mucosa. Both acute and peracute forms of the disease occur. Sudden death is associated with the peracute form of DP. The subacute form lasts 5 to 12 days. Prolapse of the penis is common in male waterfowl.

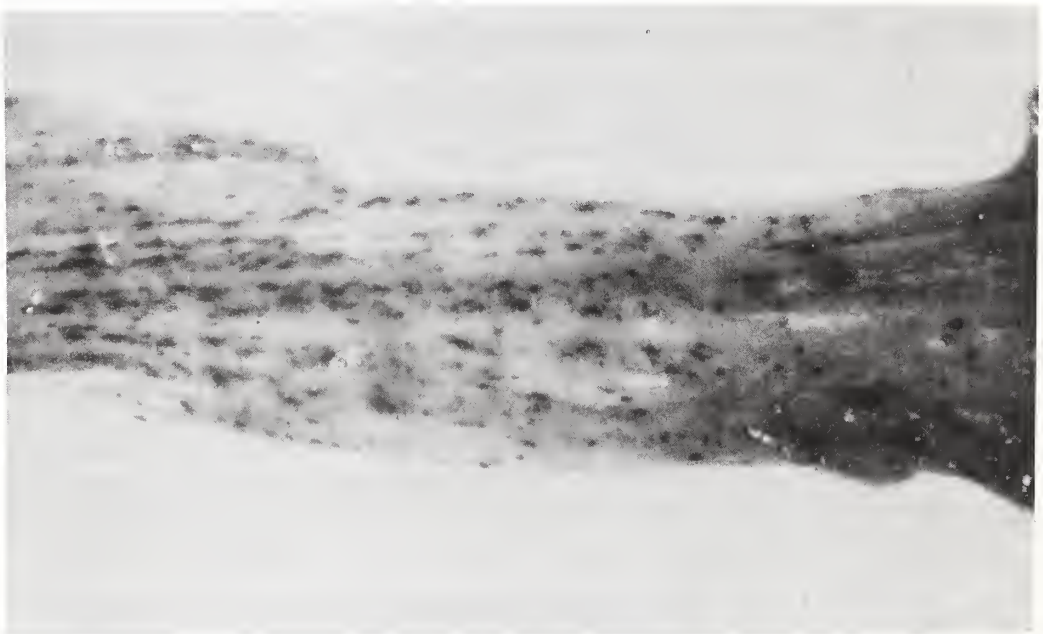
During the febrile period, the virus can be isolated from all tissues and is found in high concentrations in the esophagus, liver, and spleen. Ducks with circulating antibodies acquire immunity for life. The virus has been isolated from excretions during the course of the disease.

Infected laying ducks may have a 25- to 40-percent reduction in egg production of up to 3 weeks duration.

Gross Lesions

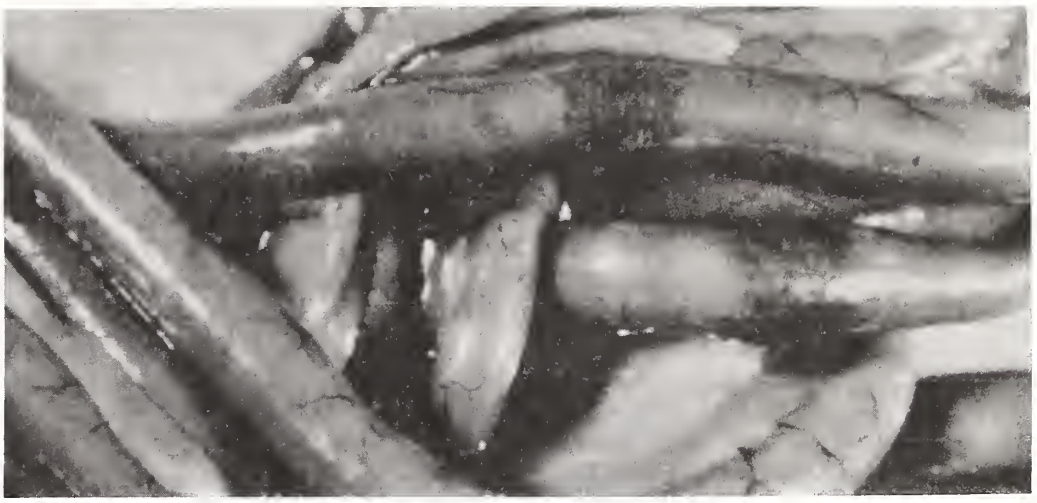
Pathological changes and mortality in DP vary among individual flocks and geographical locations. These differences may be attributed to the virus strain, route of exposure, host age, and breed or species affected. Nevertheless, certain common features are observed. These consist of progressive, exanthematous lesions of the digestive tract, hemorrhage and necrosis of visceral organs, serosal hemorrhages, and accumulation of free blood in body cavities.

Often the entire esophageal mucosa is covered by a fibrinous pseudomembrane. When the pseudomembrane is removed, submucosal petechiation may be observed along the entire length of the esophagus. The tiny papillae which are arranged symmetrically on the edge of the mucosal folds are petechiated and intensely red in color, producing a longitudinally striped appearance in immature ducks.



Linear petechiation of esophagus

Diffuse hemorrhage may be very prominent at the esophageal-proventricular junction, forming a reddish submucosal ring. The intestinal mucosa may be covered with a bloody catarrhal exudate and, on occasion, the lumen may be filled with blood. Red annular bands may appear at regular intervals within the small intestines. Location in the lymphoid tissue of the annular bands allows these submucosal hemorrhages to be seen from outside the intact intestine.



Dark red annular hemorrhagic bands in intestinal serosa

Lesions in the cloaca vary with host age and species. In young, experimentally infected birds, lesions are found in the bursa of Fabricius. The submucosa is red and edematous. Petechiae and diffuse or ecchymotic hemorrhages are visible on the mucosal surfaces.

Two types of lesions are present in the liver. In waterfowl with extensive hemorrhages and extravasation of blood in body cavities, the liver is friable, enlarged, bronze in color, and the surface is covered with pinpoint hemorrhages having a paintbrush appearance. This pattern is more noticeable in waterfowl with secondary bacterial infections. In contrast, in experimentally infected, specific pathogen-free ducks, the liver is firm and has a normal color.

In mature waterfowl, the kidneys are congested. However, in 2- to 4-week-old ducklings, there are petechial and ecchymotic hemorrhages under the capsules and throughout the renal tissues.

Focal hemorrhages are widespread. In laying ducks, the surface of the ovarian follicles contains petechial hemorrhages and/or ecchymoces, or extravasation and congestion. Occasionally, the ovarian follicle may rupture. Petechial or eccymotic hemorrhages are often present on the epicardium and endocardium, and may be seen in the fat around the coronary groove. The larynx and lungs may also contain hemorrhages.

Inoculation of DPV into 9-day-old developing embryos results in embryo death after 5 to 10 days with maximum mortality occurring about the 5th day. Bright to dark red discoloration of the embryonic tissue is observed, especially in the epidermis and musculature of the thighs and shanks. Scattered hemorrhagic foci are found in areas of the skull, neck, and muscles of the body and limbs. The embryo livers are enlarged, their surface having punctiform hemorrhagic and necrotic foci. The chorio-allantoic and yolk sac membranes are thickened, edematous, and contain diffuse or petechial hemorrhages. Similar lesions are noted in chicken embryos infected with attenuated virus. Embryos usually die 4 to 7 days after inoculation with attenuated DPV.

Histopathology

As a result of vascular damage, dependent tissues progress from hemorrhage to necrosis. Tissue destruction is noted in all lymphoid organs. These include the thymus, bursa of Fabricius, spleen, intestine (annular bands), esophageal-proventricular junction, and posterior margin of the cloaca. Within these lymphocytic organs, the lymphatic and reticular cells are destroyed. Hemorrhages and necrosis in the tissues are associated with formation of intranuclear inclusion bodies.

Serology

Serums collected from convalescent and immunized ducks contain antibodies which protect duck embryos and chicken embryos from the lethal effects of virulent and chicken embryo-adapted DPV, respectively. Virus neutralization tests may be conducted in embryos and cell cultures. Virus neutralization indices of serum from non-infected duck flocks in the Eastern, Midwestern, and Western States ranged from 1.25 $\log_{10}$ to none. In contrast, 33 to 60 percent of the serums collected from flocks 19 and 35 days after the appearance of DP had virus neutralization indices of 1.75 to 3.0 $\log_{10}$. Antigen from chorio-allantoic membranes and serums from immunized ducks were used to demonstrate precipitating antibodies, using immunodiffusion reactions.

Diagnosis

Pathognomonic esophageal lesions in waterfowl may assist in field diagnosis. However, laboratory confirmation of DP requires the isolation and identification of the virus, or the demonstration of antibody development.

Demonstration of histopathological changes in the affected organs, especially in the esophageal and cloacal linings, and in the hepatocytes are of additional diagnostic value.

Inoculation of 8-day-old duck embryos by way of the chorio-allantoic membranes was once the method of choice in primary isolations of DPV from natural outbreaks. However, it was found that duck embryos or ducklings originating from some flocks did not die after inoculation with tissue suspensions from suspected cases of DP. Therefore, simultaneous inoculation of duck embryos and week-old ducklings is recommended.

DPV may be detected by immunofluorescence and ferritin-labelled antibody methods.

A study in which chicken embryo-adapted DPV was used as an antigen in the examination of 2,000 ducks serums from commercial and wild waterfowl demonstrated that a virus neutralization index of 1.5 $\log_{10}$ or more indicates a recent infection. The use of chicken embryo-attenuated DPV and chicken embryo or cell culture systems for antibody titration is safer and more convenient than the use of DPV virulent virus and embryonated duck eggs.

Recently, an experimental reverse-passive hemagglutination test for tissues of lethally infected ducks was developed and found to be sensitive for the detection of DPV in acute infection.

Isolation of virulent DPV requires adequate specimens from at least 2 birds showing the pathognomonic esophageal and cloacal

lesions. Submission of paired serums is necessary for the demonstration of antibody development. DPV specimens must be packaged with the utmost care to preserve their integrity for virus isolation and avoid seepage of contaminated fluid from the package.

For primary isolation, chorio-allantoic inoculation is preferred and results in more rapid results than allantoic and yolk sac inoculation.

Assay of virus-neutralizing antibodies has been successful in a system using a constant dilution of reference serum (1:5) and serial 10-fold dilutions of virus.

Prevention and Control

Exposure to infection and spread of DPV may be avoided by abandoning traditional methods of commercial duck production in open waterways where domestic ducks may mix with migratory waterfowl carrying DPV.

Ducks at risk of being infected should be vaccinated. There is a need for the development of vaccines which may be applied by aerosol or in the drinking water.

Public Health

DP does not represent a hazard to humans. Infection was not reported in laboratory workers who isolated, propagated, and conducted aerosol transmission experiments and necropsies on large numbers of naturally and experimentally infected Anseriformes. (Dr. A. H. Dardiri, Plum Island Animal Disease Center, 516 323-2500)

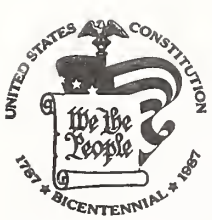
Questions about the FAD Report may be sent to:

Dr. E. I. Pilchard, Editor
APHIS, USDA, VS
Room 801, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

Thirty days before moving, send address change, and if possible, mailing label from latest issue to:

Information Management Branch
APHIS-USDA
Room G-187, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

*U.S.GOVERNMENT PRINTING OFFICE:1987-180-916:60129/APHIS


UNITED STATES CONSTITUTION
We the People
1787 * BICENTENNIAL * 1987